— MANGANESE & INFRASOUND — THE LINK TO MAD COW DISEASE

Politicians and scientists have erred in claiming that BSE is hyperinfectious and can only be managed by the wholesale slaughter of livestock, while the corporations have profited at the expense of small farmers.

Part 2 of 2

by Mark Purdey © 2002, 2003

High Barn Farm
Elworthy, Taunton, Somerset TA43PX
United Kingdom
Email: MadCowPurdey@aol.com
Website: http://www.markpurdey.com

Is Rogue Ferrimagnetic Manganese the "Infectious" TSE Agent?

Once the crucial supply of copper is curtailed in the brain, due to straightforward environmental copper deficiency or exposure to copper-chelating organophosphate (OP) insecticides, etc., the prion protein's metal bonds become vacant, rendering the protein vulnerable to bonding up with certain alternative metals such as manganese, strontium, silver or lithium. But these foreign substitutes may not act in the overall best interests of the organism, particularly if the invasive metal is in "ferrimagnetic" form.

So when ferrimagnetic manganese substitutes at the vacant copper bonds on the prion protein, the field-inducing influence of its ferrimagnetically ordered atoms will progressively corrupt the circadian-mediated pathways of electromagnetic superexchange throughout the brain, whereby a status of permanent magnetic charge is spread via a domino-style of contagious corruption which jumps from metal bond to metal bond, from prion to prion. This phenomenon is well illustrated by the classic college physics experiment, where a magnet is placed alongside a steel nail and the force field of the magnet rapidly magnetises the adjoining nail.

Once an individual's brain is contaminated by this freaky form of metamorphosed manganese, any subsequent exposure to external electromagnetic fields (e.g., UV, sound waves, radar, cell-phones, etc.) will permanently charge up the ferrimagnetically ordered manganese prions. The metals rapidly become permanently saturated with magnetic charge, generating intensive magnetic fields which, in turn, generate self-perpetuating "cluster bombs" of free-radical-mediated spongiform neurodegeneration. TSE ensues.

In this respect, the TSE-diseased brain can be likened to a solar-powered battery on continuous charge, where the manganese-loaded/copper-depleted brain is no longer equipped to deal with the incoming surges of electromagnetic energy from the external environment. Instead of utilising this energy for the body's own vital requirements, it becomes perverted into a potent force for neuronal suicide.

This theory explains why the so-called "hyperinfectious" property of the prion is a misnomer. It is the toxic ferrimagnetic metal component of the prion that serves as the so- called "infectious" pathogenic agent in TSEs (transmissable spongiform encephalopathies). So whenever scientists inoculate unfortunate laboratory animals with TSE brain tissues (e.g., tissues contaminated with this rogue manganese atom) and effectively transmit TSE, they are actually transmitting "a magnetic field–inducing capacity" that is carried along with the ferrimagnetically ordered manganese contaminant into the recipient animals which, in turn, develop TSE.

Furthermore, the concept of the rogue ferrimagnetic manganese atom as the "TSE agent" also explains why the "infectious" pathogenic capacity of the prion can survive heating to temperatures in excess of 500 degrees Celsius—since ferrimagnetic metals will hold onto their magnetic charge until they are heated to temperatures beyond their respective "curie point" temperature (e.g., 550°C for manganese 3+).

A Theory that Addresses All the Missing Links in TSE Science

Some would question how the toxic manganese theory of TSE origins can account for the well-recognised "iatrogenic" form of TSE, where growth hormone treatment of humans—which utilises pituitary tissue as the pharmacological inoculant—can lead to a form of CJD (Creutzfeldt–Jakob disease). But intriguingly, tissues such as pituitary and retina, which transmit TSE in the lab most efficiently, are the same tissues in which manganese is recognised to concentrate most intensively in the body. So once an individual is contaminated with a rogue source of ferrimagnetic manganese, any subsequent use of their pituitary tissues in pharmaceuticals for growth hormone therapy could spread the so-called "infectious" toxic agent and initiate CJD.

Others would question how this theory can account for the outbreak of the kuru strain of TSE that exclusively erupted in an isolated tribe in the Fore region of the New Guinea Highlands. The conventional dogma blames this outbreak upon the Fore tribe's traditional practice of cannibalism. Whilst cannibalism may have played a role in the bioaccumulation of manganese—particularly if the pituitary tissues were ingested in these cannibalistic binges —the fact that virtually every tribe across New Guinea had adhered to a cannibalistic lifestyle, yet remained free of kuru, needs to be addressed by those who promote this theory. And furthermore, considering that cannibalism had been traditionally practised for centuries across New Guinea, why did kuru fail to erupt until a few years after World War II?

My investigations suggest that the cause of kuru stems from the same template of eco-factors: the Fore tribe's self-sufficient lifestyle on copper-deficient soils, coupled with their scavenging of manganese-aluminium sheet metal from the fuselages of several Japanese bomber aircraft which had crashed in their area of the Highlands during World War II. The Fore folk moulded the salvaged metal to make tools, cooking pans and bowls, and these consequently contaminated their foods. They also accidentally exploded some of the bombs on board the crashed aircraft. These infamous explosions—well remembered by the surviving Fore folk—infrasonically irradiated their local environment.

This story goes on. At the mouth of the Fuji River valley in Japan is a manganese-aluminium alloy factory that manufactured these metal aircraft panels from the late 1930s and still makes Mn-Al alloy products today. The manganese-enriched chimney emissions dispersed downwind, permeating the entire length of the valley. Intriguingly, a cluster of CJD has blighted the residents of the Fuji River basin for 50 years. Note that it can take up to 20 years for the toxic effects of metal/chemical exposure to manifest.

For 18 years, my work and personal integrity have been subjected to a steady derisory trickle of ridicule and dirty tricks.

The role of prion protein genetics is also entirely compatible with the environmental facets as part of the overall multifactorial aetiology of TSEs. For it is well established that prion protein genetics plays a major role in dictating which individuals are most susceptible to TSEs—where susceptibility hinges upon the expression of a defective prion protein that can only bind two or three atoms of copper instead of the usual five. But the sole focus of TSE susceptibility studies to date has almost exclusively concentrated upon the role of prion protein genotypes. Yet my own studies have revealed that white/fair skinned or yellow/red-pigmented individuals are at much greater risk of developing TSE. This suggests that the genetics of melanin expression may also perform a genetic role in the cause of TSE.

Note that melanin is involved in cushioning the toxic side-effects of light and sound absorption. Some support for this observation was amassed when unfortunate laboratory mice were genetically engineered to express a mutant form of melanin and consequently developed spongiform encephalopathy.

Animal Pharm

Despite publication of the hard evidence in support of this theory in prestigious scientific journals (see bibliography), the various UK authorities and their incestuous clique of "minder" advisers are blindly ignoring these findings. What's more, they are doing their utmost to publicly marginalise those of us who are trying to pursue this alternative research line, and are using public money to implement their tactics of suppression in the bargain.

For 18 years, my work and personal integrity have been subjected to a steady derisory trickle of ridicule and dirty tricks. During the 1980s, my farm and family became the victims of a raft of "once in a lifetime" type of physical disasters: arson, firearm intimidation, vandalisation of my research library and communications, and an insidious infiltration by a bizarre array of bogus greens and phoney freelance journos—not to mention a seductive approach by a scantily clad pseudo student who was supposedly doing her dissertation on my theory. I became suspicious, and my investigations revealed that she was not even registered at the college where she was purportedly studying!

It invariably transpired that the true objectives of these *agents provocateurs* was to subtly set about discrediting my social and scientific esteem whilst finding out the current state of play of my research investigations. Once my work gained support from the likes of the former Defence Minister, Tom King, and HRH the Prince of Wales, the physical aspects of this harassment abruptly ceased.

My recent demands to various UK government departments—agriculture, environment, health, etc.—for access to my personal data under the new Data Protection Act revealed much of what had been going on behind the scenes. Repeated requests by Environment Minister Michael Meacher to meet personally with me had been deliberately stymied by his own officials. When Mr Meacher eventually broke through his barrage of officials to make direct arrangements with me, the meeting was postponed on five separate occasions and then arrangements completely fizzled out. Other documents revealed how the British Agrochemical Association had been organising a "joint initiative" with the Ministry of Agriculture's own grant funding department to channel public funds into a live animal trial that had been deliberately designed to refute my theory.

Since the BSE Inquiry had rejected the official scrapie/BSE hypothesis and found in favour of some aspects of my own hypothesis, the UK government responded by setting up a further mini-inquiry to re-look at the origins of BSE. The resulting publication, known as the "Gabriel Horn Report", employed a judicious selection of misrepresentation and outright bogus disinformation in order to discredit the validity of my theory.

For example, the report stated that the use of OP warblecides had ceased in the UK by 1982 and that warblecides had been used routinely on the island of Jersey. So, according to the Horn Report, if OPs were the cause of BSE, why were all of the cows that developed BSE born after 1982 and why were BSE rates so low on Jersey? Ironically, the truthful picture of the UK's compulsory twice-annual OP warblecide treatment programme was that it was *introduced* in 1982, and only *one cow* on Jersey was ever subjected to the compulsory "formal" OP warblecide treatment.

When I attempted to sue the government for defamation/loss of income resulting from the bogus statements in this globally circulated publication, it pleaded "qualified privilege" of the expert committee and then spun out the legal communications beyond the one-year post-publication mark, thereby exempting itself from my claim.

And after broadcasting of the BBC *Correspondent* film, *Mad Cows and An Englishman*, which charted my investigations, the government tried to appease the mounting public interest by inviting me to resubmit an application for funding. After sitting on my application for a year and a half, they homed in on the most fastidious, nit-picking comments

in the peer review appraisal, trumping them up as a sound scientific basis for rejection of the application. Immediately afterwards, the author of the most irrational, irrelevant critique was promoted to the government's expert TSE Surveillance Steering Committee—presumably as a reward! The UK government's tactics have thwarted the natural evolution of this promising new scientific perspective on TSEs.

A Multinational Masterplan?

The epidemiological and experimental evidence amassed to date points to the fact that TSEs are caused by a clear-cut combination of genetic and toxic environmental factors. So why do the authorities continue to treat these diseases as if they stem solely from hyperinfectious origins?

The reasons for such an irrational, Pavlovian-like stance on the part of the Establishment towards the environmental perspectives of TSEs probably hangs upon issues that are more to do with protecting academic egos, professional reputations and the vested interests of the TSE institutions and key advisers than with promoting sound scientific argument. Another reason must undoubtedly stem from the fear of massive compensation claims, should government-mandated use of OP warblecides or licensing of manganese additives be held accountable at the end of the day.

But it's imperative that we delve a bit deeper. Who are the key culprits that are currently capitalising on the fashionable scare stories which maintain that "BSE prions will exterminate us all"? Who are the people spinning out the propaganda myths that beef, lamb, venison, game and organic food (grown from animal manure) are contaminated with prions and are therefore unfit for human consumption?

The key scaremongers can invariably be traced to a mere handful of sociopathic pseudo-scientists who move between the upper echelons of government and corporation-controlled institutions. These incestuous experts are singing for their supper. They are on the payroll of the multinational chemical consortiums—corporations that have invested billions of bucks in researching and developing their genetically modified arable protein crops and the complementary package of pesticides to go with them. They have bought up oceans of acres of dirt-cheap arable land across Eastern Europe, the Third World and North/South America and they are clearly attempting to destroy anyone competing for their global protein market. Prime targets are the small mixed-livestock farming sectors of agriculture which have traditionally been the mainstay of meat and milk protein production around the world.

The multinationals' preference for mono-arable cropping land use is easily understood, since each acre of grassland that is devoted to meat and milk production requires negligible inputs of pesticide/GM seeds in relation to each acre of farmland that is devoted to agrichemical-intensive arable protein production.

Despite the scaremongering over the "hyperinfectious" nature of the prion, a basic study of the epidemiological history of TSE clearly demonstrates that this disease does *not* originate from animal-to-animal contact or through ingestion of feeds contaminated with TSE brain material.

So why do the "experts" blatantly refuse to consult the down-to-earth wisdom of the Icelandic farmers and vets who have been living with scrapie TSE for light years? When the first hint of scrapie symptoms emerge in their sheep, it is customary practice to slaughter the poor affected animal instantly, before it has had time to waste away, and eat the cooked flesh (brains and all!). And if scrapie or CWD (chronic wasting disease) can be passed on to humans via consumption, as the scientific authorities would have us believe, why have no cases of CJD erupted in these Icelandic sheep farmers? In fact, Iceland has only ever witnessed two cases of CJD in its entire medical history, and these victims had both hailed from the scrapie-free district in the far south of the country.

Despite the repeated failure of attempts to eradicate long-established TSE hotspot regions in Colorado and Iceland by enacting wholesale livestock slaughter/fallowing regimes across the cluster zones, governments are still adopting this same slaughter strategy as a first-choice means of control. But history has shown that TSEs will invariably re-erupt as soon as fresh livestock are introduced back into the slaughtered-out areas—supporting the idea that the environmental causes of TSE are still well and truly wedded to the local food chain, irrespective of the slaughter programmes.

The epidemiological and experimental evidence amassed to date points to the fact that TSEs are caused by a clear-cut combination of genetic and toxic environmental factors.

Such extreme "mammalageddon" measures do little more than remove the superficial evidence of the disease. They merely mislead the public into the illusory notion that TSE has been controlled—a good vote-catching policy for any government.

These are simple observations yet the global authorities are gripped by a manic mindset, having jumped on the assumption that TSEs stem solely from hyperinfectious origins. For example, the recent discovery of new clusters of CWD in US deer has invoked an official overreaction of unprecedented proportion. A wholesale slaughter policy of indigenous deer herds has been enacted throughout all CWD regions across the USA, leaving many of the Native American tribes without their traditional source of dietary protein. Whilst studying in Wisconsin recently, I heard the story of a deer rancher who had retained some body tissues from one of his CWD-affected deer for independent post-mortem—only to find himself subjected to a gun-point raid by wildlife officials .

These draconian slaughter measures are invariably promoted by the same hard-core cell of "expert" global advisers—the hysterics who dreamed up the hyperinfectious hypothesis in the first instance. By insisting on the burial or incineration of the evidence of their own control measures—for example, the thousands of carcasses of slaughtered animals—and then enforcing the fallowing of the land, the experts are placing themselves in a foolproof position where the success or failure of their control measures can never be properly assessed. In this respect, they can guarantee keeping their professional reputations afloat for the remaining lifespan of their careers.

But who is questioning the scientific reasoning for executing this final farcical solution on these poor creatures? The unilateral adoption of a policy of "totalitarian overkill" of a few million healthy animals across the world has been received with almost complacent acceptance. Such perverse and senseless "carry-ons" have sadly become the daily "non-stories" of our modern times.

Reports pop up with ever-increasing frequency of so-called TSE precautionary control programmes being enacted after 1% or more animals in a flock or herd prove positive to the TSE genotype test—an endemic phenomenon that has existed for light years without ill effect. Among these control programmes were the annihilation of a herd of water buffalo in Vancouver and sheep flocks in Vermont, the slaughter of 400,000 cows in Germany and the erasure of thousands of scrapie-susceptible traditional sheep flocks and goat herds from the European hillsides—all healthy animals.

Along with the sad threat to the survival of some indigenous wild and domestic animal breeds, there is the threat of losing their valuable outputs of manure—the heartbeat of humus supply which protects the soil against the erosive forces of nature and feeds the fertility of the earth that ultimately sustains all life on the planet.

Furthermore, these slaughter measures are imposing the death knell on the survival of traditional peasant cultures—lifestyles which have evolved to be symbiotically dependent upon their livestock enterprises and the income they generate. We are saying farewell to one of the last bastions of our cultural identity: an holistic charm that flavours the landscapes which have been etched out by centuries of occupation under peasant family farms. That delicate, ethereal relationship that flows between the soil, crops, livestock and landscape is under threat, and along with it the aesthetically pleasing array of idiosyncrasies that go hand in hand with peasant lifestyles: the architecture, craft skills, folklore and dialects that have set the rich rustic outbacks apart from the homogenised synthetica of the city.

The all-out slaughter tactic betrays a total lack of interest in the cause, prevention or cure of this grotesque disease. The Establishment's current global agenda (*Agenda 2000*) to depopulate livestock numbers at whatever the cost is for reasons that have nothing whatsoever to do with (illusory) health risks to the human race, but more to do with envisioned profits from multinationally sourced GE/GM proteins.

I cannot help but feel that the global leaders have sold out to the multinational carrot. PR tactics used to promote "important" government policy increasingly capitalises upon some emotive scare story as a means of manipulating public mentality into conforming with the overall global agenda of the corporations. In much the same way as a war for Iraqi oil has been presented under the pretext of a morally justified war to rid Iraq of weapons of mass destruction, so the corporations' war to rid the world of livestock protein has been presented under the guise of ridding the world of the "health risks" posed by hyperinfectious prions.

This story returns us to the lessons that can be learned from the intuitive wisdoms of the people on the ground.

The Broader Picture

The BSE debacle represents the mere tip of the iceberg of Establishment ineptitude and socio-eco-irresponsibility. It displays a clear-cut example of the far-reaching extent to which the talons of multinational monopolies can stretch to protect their global master-plan for the rapidly expanding "health and food-chain industry". Can we afford to allow this insidious mode of food-chain control to continue unabated and unregulated?

There is an increasing groundswell of public unease concerning our polluted environment and its unknown effects on our health and long-term survival. Mounting public suspicion is making transparent the array of so-called independent scientific experts and medical spin-doctors who are called to advise governments and address the public on all aspects of the impact of chemical, metal, radioactive and electromagnetic pollutants upon our food chain.

This story returns us to the lessons that can be learned from the intuitive wisdoms of the people on the ground. At the same time it alerts us to the insidious and unscientific techniques which the incestuous clique of official "experts" employs to marginalise and discredit those who dissent from the totalitarian line. It shows us the ill-conceived basis upon which the Establishment's positions are truly founded, as well as the woeful degree of administrative complacency over issues which in most cases are matters of life and death for normal people.

Dispatches from the Front Line

My most recent eco-detective adventures across vCJD cluster zones in Japan, the USA, Sardinia and the UK involved my prospecting for a broader range of metals in TSE environments than I'd previously sought.

The analytical results have thrown up some interesting new possibilities in the quest for understanding the true causes of TSEs, unearthing high levels of the ferrimagnetic metal strontium as well as the usual high levels of manganese in the TSE environments, but normal levels in the adjoining TSE-free areas.

Much like manganese, strontium is emitted as a significant contaminant from volcanoes as well as from factories refining metals/steel or producing glass/dyes/explosives/paints/fireworks. It is used in surgery/dentistry, and as a bone/antler promoter in mineral supplements for humans and deer, etc. Strontium also competes with and replaces vacant calcium, magnesium and copper sites on proteins in the biosystem, so tests are currently in place to see if this metal can bind to copper-deprived prion protein like manganese.

The strontium facet offers a promising new theoretical possibility that can be considered without disturbing the basic pathogenic template of this TSE causal theory—where the rogue ferrimagnetic strontium substitutes at the copper-depleted metal bonds on the prion protein, thereby impairing the protein's ability to conduct the vital "life force" electromagnetic energies derived from incoming light and sound.

It naturally follows that exposures to high levels of naturally occurring strontium could have triggered off the *traditional* strains of TSE, whereas exposures to the more reactive, radioactive strontium-90 could have caused the more aggressive, *new variant* strains of TSE in younger mammals.

Perhaps the strontium-90 emissions from the April 1986 Chernobyl nuclear disaster—the bulk of which at that time were deposited by the substantial rainstorms over northwestern Europe (e.g., UK, Ireland, Britanny)—were responsible for setting up susceptibility for mad cow disease in any cattle, humans or cats that had been simultaneously exposed to the copper-chelating, organo-dithiophosphate warble fly/headlice insecticides. The first reported case of BSE erupted in October 1986, whereas the almighty BSE epidemic that followed was largely contained within the key Chernobyl fallout zone.

It seems that several species of rogue ferrimagnetic metal—be it manganese, strontium or bismuth—may individually carry the potential to act as the TSE trigger in the copper-depleted brain. The resulting

Continued on page 77

Manganese & Infrasound: The Link to Mad Cow Disease

Continued from page 36

mineral imbalance compromises the brain's ability to deal with low-frequency infrasonic shocks.

In part one, we saw how pockets of intensive infrasound can be geographically correlated with the TSE cluster locations. Recent research conducted by Mariana Alves-Pereira (Department of Environmental Science, New University of Lisbon, Caparica, Portugal) has identified the symptoms and biochemistry of low-frequency noise damage, where the symptom profile—involving hypersensitivity to sound, reclusive behaviour, aggression, paranoia, cardiac complications, etc.—is near identical to some of the clinical features of TSEs. Furthermore, the well-known metabolic association between the healthy prion protein and the actin and melanin proteins, plus the fact that actin and melanin play a crucial role in the absorption of incoming vibro-acoustic waves, may offer support for the suggestion that TSEs result from a metal-deformed prion protein and the subsequent breakdown in the brain's ability to deal with low-frequency noise.

Governments and corporations have deliberately conspired to manipulate what the public get to hear surrounding the causes of TSE—not to mention the causes of so many other modern ailments. Their concealment of the whole truth is betrayed by the fact that the UK's BSE Inquiry team was debarred from accessing 30% of the government's data on BSE—because it was "classified" under the Official Secrets Act.

Never before in the history of human medicine has the populace on a global scale been so successfully indoctrinated by the healthcare propaganda of the multinational and governmental lobbyists. A mere handful of ministers and corporate-controlled advisers have served as traitors to their own people and planet. Their deviant behaviour should be treated as criminal negligence. ∞

Bibliography

NB: For additional references, refer to the "Cattle Practice" article on Mark Purdey's website, http://www.markpurdey.com.

• Purdey M. Ecosystems supporting clusters of sporadic TSEs demonstrate excesses of the radical-generating divalent cation manganese and deficiencies of antioxidant co-factors Cu, Se, Fe, Zn. Does a foreign cation substitution at Prp's Cu domain initiate TSE? *Medical Hypotheses* 2000 54(2):278-306.

• Purdey M. Does an ultraviolet photo-oxidisation of the manganese-loaded/copper-depleted prion protein in the retina initiate the pathogenesis of TSE? *Medical Hypotheses* 2001 57(1):29-45.

• Purdey M. The Mn-loaded/Cu-depleted bovine brain fails to neutralise incoming shock-bursts of low-frequency infrasound. The origins of BSE? *Cattle Practice* 2002 (Oct) 10(4):311-325.

• Brown D, Hafiz F, Glassmith L, et al. Consequences of manganese replacement of copper for prion protein function and proteinase resistance. *EMBO J* 2000 19(6):1180-1186.

• Wong BS, Chen SG, Colucci M, Xie Z, Pan T, Liu T, Sy MS, Gambetti P, Brown DR. Aberrant metal binding by prion protein in human prion disease. *J Neurochem* 2001 78:1400-1408.

• Gordon I, Abdulla EM, Campbell IC, Whatley SA. Phosmet induces up-regulation of surface levels of cellular prion protein. *Neuroreport* 1998 9(7):1391-1395.

About the Author:

Mark Purdey is a traditional mixed family farmer in the UK who successfully defeated the UK government's compulsory insecticide treatment order in the High Court of London in 1984. He writes, lectures and broadcasts on environmental health issues, whilst pioneering global ecodetective investigations into the causes of brain disorders, such as BSE. Mark's analytical studies have identified some "common toxic denominators" involved in the cause of BSE, and his published hypothesis is currently gaining support from studies conducted at US, Japanese and European universities.

The full text of Mark's article can be found on his website, http://www.markpurdey.com. It was originally published as "Educating Rida"—"Rida" being the Icelandic term for transmissible spongiform disease (TSE).